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Issue Date: 16 October 2002

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In the Matter of

BETTY BAILEY, widow of THOMAS BAILEY
Claimant

v.

CONSOLIDATION COAL COMPANY
Employer

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party-in-Interest
.....

Case No: 1998-BLA-00825

DECISION AND ORDER ON REMAND

On December 7, 1999, I issued a Decision and Order in the above-captioned case, awarding benefits. I found that the Claimant is an eligible survivor of the Miner, that her survivor's claim was filed on May 12, 1997, that Part 718 of the Regulations applied, and that the Miner had thirty years of coal mine employment. As to the issue of pneumoconiosis, I first found that the doctrine of collateral estoppel did not preclude my consideration of whether the Miner suffered from pneumoconiosis.[1] I then found that:

> There is a substantial amount of conflicting evidence in the record, with x-ray and CT scan evidence interpreted by experts as both finding and ruling out the presence of pneumoconiosis. I give more weight to the reports from the four examining physicians, as they would have first hand knowledge of the Miner's condition. See Grigg v. Director, OWCP, 28 F.3d 416 (4th Cir. 1994); Adkins v. Director, OWCP, 958 F.2d 49 (4th Cir. 1992).

[1] The Miner filed a claim for benefits on March 12, 1987. In a Decision and Order dated June 16, 1989, and a subsequent Decision and Order on Remand dated August 6, 1991, Administrative Law Judge Clement J. Kichuk awarded benefits. The employer had stipulated to the presence of pneumoconiosis. The miner's claim was in payment status when he died. (DX 18).

> Of the examining physicians, only Dr. Vasudevan in his April 1, 1987 examination failed to diagnose pneumoconiosis (DX 18). Drs. Qazi, Abernathy, and Hatahet all diagnosed pneumoconiosis and related it to the Miner's multiple years of exposure to coal mine dust (CX 1; DX 18). I give the greatest weight to Dr. Hatahet's report because he was the Miner's treating physician for a period up to and including his death in 1997, and because his report is supported by two other examining physicians, the death certificate filled out by the attending physician Dr. Khokar, the 1988 consultation reports by Drs. Fino and Morgan, and the 1998 reports of Drs. Dahhan and Gaziano (CX 1; DX 9, 18; EX 5). See Clark v. Karst-Robbins Coal Corp., 12 B.L.R. 1-149 (1989)(en banc); Gillespie v. Badger Coal Co., 7 B.L.R. 1-839 (1985). I give less weight to Dr. Vasudevan's report because it is outdated, and inconsistent with weight of the medical evidence available to him in 1987. See Clark, supra. I give less weight to the 1998 consultative reports of Dr. Fino, because he contradicts his 1988 report, and he did not examine the Miner (EX 2, 3). I give less weight to the 1988 consultative reports of Drs. Castle, because, he did not examine the Miner (EX 1, 4). See Bogan v. Consolidation Coal Co., 6 B.L.R. 1-1000 (1984). Accordingly, I find the existence of pneumoconiosis has been established pursuant to §718.202(a)(4).

I then found that the Miner's death was due to pneumoconiosis. §718.205(c)(1), (2). Benefits were awarded commencing May 1, 1997.

The Employer appealed that award to the Benefits Review Board ("the Board"). On January 31, 2001, the Board vacated the award and remanded the case for further consideration consistent with its opinion. The Board found that the doctrine of collateral estoppel did not apply. However, it vacated the findings of pneumoconiosis and death due to pneumoconiosis. The other findings were unchallenged on appeal. Following the remand, I issued an Order on August 20, 2001, granting the parties thirty days in which to file any additional comments.

Evidence

The death certificate states that the Miner died on May 5, 1997 at the age of sixty-one. The immediate causes of death were bilateral pneumonia, complicated pneumoconiosis, and massive pulmonary fibrosis. An autopsy was not performed. The certifier was Dr. Muhammed Khokar. (DX 5).

The following is a summary of the pertinent medical evidence of record:

A. Chest X-rays

Ex.No.	Date of X-ray	Film Qual.	Physician/ Qualifications[2]	Interpretation
DX 17	3/15/86	2	Wheeler/BCR, B	Probable subtle 3 cm mass or adenopathy lower right hilum and partly rounded 2x4 cm mass in anterior inferior right or LLL involving lower oblique fissure compatible with inflammatory disease or tumors. Subtle increased lung markings RML compatible with early interstitial fibrosis or infiltrate. Moderate discoid atelectasis RLL and minimal discoid atelectasis LML. Focal fibrosis right apex from healed tuberculosis.
EX 1	3/15/86	2	Castle/B	Linear atelectasis or scar LLZ and LMZ.
EX 2	3/15/86	1	Fino/B	0/0. Diffuse alveolar filling disease process.
EX 5	3/15/86	1	Dahhan/B	2/1, p/p, six zones. Disc like atelectasis.
DX 18	4/1/87	1	Aycoth/BCR, B	1/0, p/s, six zones.
DX 18	4/1/87	1	Gaziano/B	1/1, p, six zones. Bilateral linear

[2] The symbol "BCR" denotes a physician who has been certified in radiology or diagnostic roentgenology by the American Board of Radiology, Inc. or the American Osteopathic Association. 20 C.F.R. § 727.206(b)(2).

The symbol "B" denotes a physician who was an approved "B-reader" at the time of the x-ray reading. A B-reader is a physician who has demonstrated expertise in assessing and classifying x-ray evidence of pneumoconiosis. These physicians have been approved as proficient readers by the National Institute of Occupational Safety & Health, U.S. Public Health Service pursuant to 42 C.F.R. § 37.51 (1982).

				atelectasis.
DX 18	9/10/87	-	Pope	Overexpanded with bullous change. Prominent reticulonodular pattern in upper zones compatible with pneumoconiosis. Some scarring left base.
EX 1	9/10/87	-	Castle/B	Fine linear infiltrates with linear scars. Probably granulomatous disease, possibly sarcoidosis.
EX 2	9/10/87	1	Fino/B	0/0. Diffuse alveolar filling disease process.
EX 5	9/10/87	1	Dahhan/B	2/1, p, six zones. Bilateral atelectasis lower zones.
DX 17	6/9/92	2	Wheeler/BCR, B	Oval mass or loculated interlobar effusion in lower right hilum and lower right oblique fissure compatible with inflammatory disease or tumor. Subtle increased lung markings in mid lungs compatible with subtle linear interstitial fibrosis or lymphatic spread of tumor. 1.5 cm partly rounded radiodensity lower right apex compatible with granuloma or tumor. Moderate to marked discoid atelectasis in lower lobes.
EX 1	6/9/92	-	Castle/B	Fine linear bilateral infiltrates with linear bands extending from each hilum. Probably hilar adenopathy. Granulomatous disease; possible sarcoidosis.
EX 2	6/9/92	1	Fino/B	0/0. Diffuse alveolar filling disease process.
EX 5	6/9/92	1	Dahhan/B	2/1, p, six zones. Atelectasis. Cancer?

DX 17	10/12/93	1	Wheeler/BCR, B	5x7 cm mass RLL and lower right hilum and ill defined mass or loculated interlobar effusion right oblique fissure compatible with inflammatory disease or tumor. Marked discoid atelectasis lower lobes. Probable subtle nonspecific linear interstitial fibrosis RML. Subtle nodule or scar in lower right apex compatible with TB unknown activity, probably healed.
EX 1	10/12/93	-	Castle/B	Fine linear bilateral infiltrates with linear bands extending from each hilum. Bilateral hilar adenopathy. Possible mass right hilum. These changes are compatible with sarcoidosis, granulomatous disease.
EX 2	10/12/93	1	Fino/B	0/0. Diffuse alveolar filling disease process.
EX 5	10/12/93	1	Dahhan/B	1/1, p/q, upper and mid zones. Atelectasis.
DX 17	3/18/94	2	Wheeler/BCR, B	5x7 cm mass lower right hilum and RLL compatible with inflammatory disease or tumor. Interlobar effusion or mass in lower right oblique fissure. Subtle nonspecific linear interstitial fibrosis in mid lungs or lumphatic spread of tumor. Small nodule lower right apex compatible with granuloma from healed TB.
EX 1	3/18/94	1	Castle/B	Fine linear bilateral infiltrates with bands extending out from the hila. Hila adenopathy with possible mass right hilum. Changes are compatible with

				sarcoidosis, granulomatous disease.
EX 2	3/18/94	1	Fino/B	0/0. Diffuse alveolar filling disease process.
EX 5	3/18/94	1	Dahhan/B	1/2, p, six zones. Ca. Co. Ih. Atelectasis.
DX 17	4/23/96	2	Wheeler/BCR, B	No change from 3/18/94.
EX 1	4/23/96	2	Castle/B	Bilateral fine linear infiltrates with scars extending from each hilum. Possible mass right hilum. Hilar adenopathy. Changes are compatible with sarcoidosis, granulomatous disease.
EX 2	4/23/96	1	Fino/B	0/0. Diffuse alveolar filling disease process.
EX 5	4/23/96	1	Dahhan/B	2/1, p, upper and mid zones. Ca. Di. Ih. Atelectasis.
DX 17	12/11/96	2	Wheeler/BCR, B	5-6 cm mass in lower right hilum and RLL compatible with conglomerate TB and small granuloma in lower right apex largely hidden by clavicle due to healed TB. Minimal nonspecific linear interstitial fibrosis in right mid lung. Minimal bilateral discoid atelectasis mid lungs. Probable minimal emphysema. No evidence of silicosis or CWP.
EX 1	12/11/96	U/R	Castle/B	Unreadable.
EX 2	12/11/96	1	Fino/B	0/0. Diffuse alveolar filling disease process.
EX 5	12/11/96	3	Dahhan/B	1/1, p, upper and mid zones. Right hilar mass. Ca.
CX 1	4/21/97	-	Shahan	Chronic severe interstitial disease and bilateral perihilar soft tissue

				masses which are unchanged from multiple prior examinations. Right midlung pneumonia, unchanged.
EX 1	4/21/97	2	Castle/B	Fine linear infiltrates bilaterally. Scars extending from both hila. Hilar adenopathy. Changes are compatible with sarcoidosis, granulomatous disease. Possible right hilar mass.
EX 2	4/21/97	1	Fino/B	0/0. Diffuse alveolar filling disease process.
CX 1	4/24/97	-	Shahan	Chronic severe interstitial disease with a right perihilar soft tissue mass. Right mid lung pneumonia, unchanged.
CX 1	4/25/97	-	Shahan	Patchy infiltrate in RML may have improved slightly since recent exams; this is difficult to evaluate due to underlying chronic severe interstitial disease and a right perihilar conglomerate mass.
CX 1	4/29/97	-	Fowler	No change in right midlung pneumonia, chronic interstitial fibrotic scarring, and COPD since 04/25/97.
EX 1	4/29/97	2	Castle/B	Fine bilateral linear infiltrates. Linear scars from each hila. Hilar adenopathy. Possible right hilar mass. Changes are compatible with sarcoidosis, granulomatous disease.
EX 2	4/29/97	1	Fino/B	0/0. Diffuse alveolar filling disease process.
EX 5	4/29/97	2	Dahhan/B	1/2, q/p, six zones. Ax? Right hilar mass. Ca. Atelectasis.

CX 1	5/2/97	-	Rao	Persistent right midlung pneumonia and bilateral conglomerate masses of pneumoconiosis. Since 4/29/97, the interstitial abnormalities have increased suggesting superimposed congestive heart failure.
CX 1	5/3/97	-	Rao	Pneumoconiosis with conglomerate densities. Superimposed interstitial abnormalities suggesting congestive heart failure appear to have improved.
CX 1	5/4/97 (2)	-	Rao	Since 05/02/97 there has been an increase in the infiltrates in the RUL and RLL. Severe underlying chronic interstitial lung disease precludes differentiation of minor differences in the rest of the lungs. Conglomerate masses are also seen, especially in the perihilar regions.

B. CT Scans

At the time he reviewed the above noted x-rays on March 19, 1998, Dr. Paul S. Wheeler also reviewed the CT scans obtained on October 13, 1993, June 14, 1995, and April 24, 1996. His interpretation was mass in lower right hilum and RLL with tiny calcifications in its lateral portion compatible with conglomerate tuberculosis and small calcified granuloma from healed tuberculosis; mass in lower left oblique fissure and mass in medial portion LLL below hilum compatible with inflammatory disease; minimal nonspecific linear interstitial fibrosis mainly in RML and lateral periphery LML and few tiny nodules in periphery LLL and pleura compatible with granulomata; discoid atelectasis in both lungs; and minimal emphysema in lower lungs. Dr. Wheeler could not rule out cancer in this case and suggested a biopsy. (DX 17).

Dr. James R. Castle reviewed the three CT scans and concluded that they did not show changes consistent with coal workers' pneumoconiosis. He stated that:

> These scans show changes consistent with either a granulomatous process such as sarcoidosis, other granulomatous disease, or the possibility of tumor exists. There are several masses in both lungs in the

> lower to mid lung zones associated with some interstitial changes that are linear and irregular. The upper lung zones are not characterized by the presence of small round regular opacities. It is my opinion that these changes are not associated with CWP but rather represent some other process such as a granulomatous process such as sarcoidosis.

(EX 1).

Dr. Gregory J. Fino interpreted the three CT scans as negative for pneumoconiosis with no rounded opacities in the upper lung zones. He found enlargement of the hilar lymph glands and diffuse interstitial changes. He felt the changes and progression over time was consistent with sarcoidosis. Another possibility was pulmonary alveolar proteinosis. (EX 2).

C. Medical Opinions

The Miner was examined by Dr. C.P. Vasudevan on April 1, 1987. He noted the Miner's employment history, family history, individual health history, smoking history (never smoked), and present complaints. He diagnosed hypertension and noted the positive Category 1 X-ray interpretation of Dr. Aycoth. A pulmonary function study revealed a mild restriction. An arterial blood gas test at rest showed moderate hypoxemia. A limited stress test showed "moderate reduction in the exercise capacity due to non-cardiac and non-ventilatory causes as the patient did not reach his target heart rate or ventilatory limitation. No evidence of exercise induced hypoxemia." Dr. Vasudevan made no express statement about the extent of the Miner's disability due to lung impairment. (DX 18).

Dr. Albert Abernathy examined the Miner on September 10, 1987 on behalf of the Employer. He also noted the Miner's histories. The Miner was using medication prescribed by his physician for breathing. The physical examination did not reveal any abnormalities of the heart or lungs. A walking test showed the presence of exercise asthma. The Miner also undertook the Bruce Protocol exercise, but was discontinued at the end of the first stage due to marked weakness. An X-ray reading by Dr. Pope was positive for over expansion and pneumoconiosis. A pulmonary function study revealed restrictive ventilation with obstruction to airflow small airways. An arterial blood gas test revealed initial values of pCO2 30/pO 69; resting pCO2 27/pO2 74; and exercise pCO2 28/pO2 63. The overall evaluation of exercise was "poor tolerance to exercise - not able to do vigorous work." Dr. Abernathy's impression was coal workers' pneumoconiosis and bullous emphysema. He stated that:

> [I]t appears that he has severe pulmonary disease as reflected by desaturation of the blood on exercise. He has very poor exercise tolerance with minimal oxygen uptake on exercise. He evidently is not able to

> undertake any vigorous occupation though he may be able to do sedentary work. The source of the bullous emphysema is not readily determined but since he has never smoked cigarettes it must be assumed that this is related to his occupational history of exposure to coal dust.Dr. Abernathy is board-certified in internal medicine. (DX 18).

Dr. Naeem A. Qazi interviewed and examined the Miner on May 3, 1988. He also reviewed medical records from 1986-87. Physical examination revealed wheezing, diffused bilateral rhonchi, and decreased breath sounds. Dr. Qazi concluded that:

> Mr. Bailey has several components in his clinical history and on his evaluation by lab to suggest significant degree of chronic obstructive pulmonary disease secondary to his occupational exposure. His chest x-ray shows diffused nodular density measuring 2-5 cm. On clinical examination, he does have dyspnea and wheezing and has been gradually getting more and more dyspneic on minimal exertion. He does have resting hypoxia of a moderate degree with a pO2 of 64 to 68mm on various blood gases and in the absence of smoking, this is significant and without any other cause, it has to be directly related to his interstitial pneumoconiosis. His pulmonary function studies also show some degree of diffused airway obstruction occuring primarily in the peripheral smaller and larger airways and again, in the absence of smoking and any other pulmonary disease, this has to be directly linked to his interstitial pneumoconiosis.

(DX 18).

Dr. W.K.C. Morgan reviewed medical records and issued a report on August 17, 1988. He concluded that the pulmonary function study obtained by Dr. Vasudevan did not suggest any significant obstruction and, if valid, showed minimal restriction. He noted that the exercise test results indicated submaximal effort who was capable of exercising to a greater extent. As to the blood gas studies, he noted that Dr. Zaldivar found them to be valid and that "the blood gas values at rest showed a pO2 65 mm and a pCO2 of 35 mm. These would indicate mild hypoxemia." Dr. Morgan found Dr. Abernathy's pulmonary function study invalid. The arterial blood gas test showed mild hypoxemia. Dr. Morgan observed Dr. Qazi's diagnosis of COPD and found it to be incorrect in that "if there is any impairment, it is restrictive in nature." Dr. Morgan concluded that the Miner had a normal or near normal ventilatory capacity, with only some possible restriction. As for the Miner's blood gases, he stated that:

> Mr. Bailey has mild hypoxemia ... I am not sure of the cause of this, but it certainly is not related to a decrement in ventilatory capacity. It may be a

> consequence of ventilation perfusion mismatching due to some other condition. ... It is impossible to be certain of the cause of his mild hypoxemia but I do not believe that the minor blood gas abnormalities would impair his capacity to work. The cause of the reduced diffusing capacity and hypoxemia is almost certainly some intrinsic lung disease, not occupational in origin.

Dr. Morgan accepted interpretations of Drs. Vasudevan and Gaziano that the Miner had coal workers' pneumoconiosis. (DX 18).

Dr. Gregory J. Fino also performed a record review on behalf of the Employer and issued a report on August 18, 1988. He found a very mild restrictive ventilatory defect and mild hypoxia from Dr. Vasudevan's tests. He also found that the exercise study demonstrated submaximal effort. The chest x-ray reading by Dr. Pope as showing "bullous change" was challenged by Dr. Fino as was Dr. Abernathy's diagnosis of bullous emphysema on the basis of the x-ray reading. Dr. Fino countered by explaining that none of the x-ray readings of the chest x-ray performed on April 1, 1987 noted bullous changes and bullous emphysema is not a complication of simple coal workers' pneumoconiosis. He concluded by saying that "although this man could possibly have bullae on the chest x-ray, the physiologic studies performed by Dr. Abernathy do not show that this bullous emphysema if present has caused any respiratory impairment." Dr. Fino found the November 11, 1987 pulmonary function study to be invalid and disputed Dr. Qazi's diagnosis of obstruction therefrom. He concluded that the Miner probably had coal workers' pneumoconiosis based on the x-ray readings; however, he did not relate the restriction to the Category 1 pneumoconiosis because such a relationship would be unusual. Dr. Fino stated that Dr. Abernathy's diagnosis of exercise induced asthma may be correct and should be evaluated. (DX 18). Dr. Fino is board-certified in internal and pulmonary medicine. (DX 18; EX 4).

Dr. Muhammed I. Khokar's office notes indicate that he examined and treated the Miner on numerous occasions from October 19, 1993 through April 8, 1997. The Miner had continuing diagnoses of COPD, complicated pneumoconiosis, and massive pulmonary fibrosis. His Theophylline level was routinely checked. His placement on steroids to treat the COPD caused side-effects. The Miner was placed on oxygen in addition to the bronchodilators. (CX 1). Dr. Khokar is board-certified in internal medicine. (CX 2).

The records of Bluefield Regional Medical Center show that the Miner was admitted on December 4, 1996 on an emergency basis due to severe respiratory distress. Dr. Radha K. Krishnan saw him in consultation on December 4 and 11, 1996 for pulmonary management. Dr. Krishnan was informed of the Miner's history of COPD and CWP, and also that the Miner "ha[d] been thoroughly investigated for possible lung CA and this was never found." Examination revealed chest remained tight with increased anterior and posterior diameter of the chest; flat diaphragms; markedly distant and diminished breath sounds; some rales over both lower lobes posteriorly and

laterally, appeared to be from pulmonary fibrosis; and indrawing of intercostal spaces bilaterally. Tests and treatment were undertaken. Dr Krishnan's diagnoses were acute and chronic respiratory failure, with acute respiratory distress; COPD, CWP with acute exacerbation, in pulmonary, cause not apparent; pulmonary sepsis; and hypotension, cause not apparent, possibly related to pulmonary sepsis, and septic shock. (DX 9).

Martin Sherer, D.O., treated the Miner on April 7, 1997. The doctor noted that the Miner was incapacitated from advanced obstructive lung disease, and oxygen dependent. The diagnoses were acute purulent bronchitis and advanced obstructive lung disease. (DX 8).

A ventilation scan on April 29, 1997 was interpreted by Dr. D. Fowler as showing severe obstructive airway disease bilaterally, and a perfusion scan as showing intermediate probability of pulmonary embolic disease. (CX 1).

On April 30, 1997, a bilateral venous doppler sonography of the right and left lower extremities was negative. (CX 1).

On September 16, 1997, the OWCP requested Dr. Dominic Gaziano, who is board-certified in internal and pulmonary medicine, to review the medical records and give an opinion. He responded that:

> While the death certificate states there was complicated CWP - none of the x-ray (B read) reports have indicated such was present. The official B reading x-rays are about 10 years old and new reading is not in record. He died with CWP and died of a respiratory illness - I believe CWP was a significant contributing factor in his death.

(DX 9).

On December 9, 1997, Dr. Yasir Hatahet wrote that:

> Mr. Bailey expired at Bluefield Regional this past May, and his death is clearly related to preexisting coal worker pneumoconiosis. The patient had very little functional lung tissue to start, because of advanced coal dust induced pulmonary fibrosis. During his terminal illness, the patient acquired pneumonia in one lung, and had documented pulmonary emboli in the other, thus obliterating all useable functional lung. Had it not been for the preexistent lung disease (CWP), the patient would have been able to cope with his acute illness much better. He might have even survived had he not had preexisting lung disease.

(CX 1). Dr. Hatahet is board-certified in internal medicine. (CX 3).

Dr. James R. Castle reviewed medical records on behalf of the Employer and issued a report on June 3, 1998. He concluded that the Miner did not have coal workers' pneumoconiosis because he did not have the physical findings (consistent findings of rales, crackles, or crepitations), the X-ray evidence (adopting Dr. Wheeler's findings in addition to considering others' and his own), nor any significant respiratory impairment. He noted that no accurate exercise blood gas test was obtained. Dr. Castle attributed the Miner's death to the bacterial pneumonia, in this case, of the type Serratia. He concluded that the Miner "had evidence of either sarcoidosis or old healed granulomatous disease which resulted in multiple scars and abnormalities in his chest x-rays and CT scans." (EX 1).

Dr. Fino issued a supplemental report on July 21, 1998 after reviewing additional medical records and considering his own x-ray and CT scan readings. He concluded that:

> There was no doubt that this man had acute and chronic respiratory disease and acute and chronic respiratory failure.
>
> However, I do not find any evidence based on my review of the information that stated that this was related to a coal mine dust related disease. He had either sarcoidosis or diffuse interstitial pulmonary fibrosis. The other possibility that would have to be entertained is a granulomatous infection.
>
> I would amend my previous report and state that this man did not have a coal mine dust related pulmonary condition and did not have coal workers' pneumoconiosis.
>
> I believe that there was a disabling respiratory impairment primarily of gas transfer noted in the past which was unrelated to the inhalation of coal mine dust. It was clearly related to this man's underlying interstitial lung disease. As stated earlier, that interstitial lung disease was not due to coal mine dust inhalation.
>
> His death was respiratory in nature, but his death was not caused, contributed to or hastened by the inhalation of coal mine dust.
>
> Finally, I would like to comment on the diagnosis of complicated pneumoconiosis. Complicated pneumoconiosis is a condition that occurs on a background of simple pneumoconiosis. It is defined as rounded opacities in the upper middle lung zones greater than 1 cm in diameter.
>
> This man did not have complicated pneumoconiosis. First of all, I did not find a background of simple

> pneumoconiosis. Secondly, that area that was affected with these masses on his CT scan was in the lower lung zones. The CT scan ruled out complicated pneumoconiosis, therefore, I do not agree with that diagnosis.

(EX 2).

Dr. Fino issued another report on August 12, 1998 after reviewing additional medical records, including Dr. Khokar's office notes, positive X-ray readings, and Dr. Hatahet's letter. His opinions remained the same. Dr. Fino pointed out that "the use of bronchodilators is not consistent with treatment of a coal mine dust-related lung condition" and that "there is no good clinical evidence in the medical literature that coal mine dust inhalation in and of itself causes significant obstructive lung disease irrespective of its ability to be reversed following bronchodilators." (EX 2).

Dr. Castle also issued a supplemental report on August 12, 1998 after reviewing additional medical records. His opinions also remained the same. He stated that:

> The radiographic changes that are present are not those seen with coal workers' pneumoconiosis regardless of the previous statements in this current data set. Coal workers' pneumoconiosis does not cause the presence of irregular opacities in the lower lung zones associated with hilar node enlargement.
>
> I would disagree with Dr. Hatahet's conclusions for several reasons. As noted previously, after leaving the mines he did not have evidence of a disabling respiratory condition manifested by significant physiologic abnormalities. His physiologic abnormalities were very minimal after leaving the mines as documented by the pulmonary function study of Dr. Abernathy in 1987. These data were above federal disability standards. Even though he had an abnormal chest x-ray, these findings were not due to coal workers' pneumoconiosis. He also died as a result of a very severe pneumonic process which occurs in the general public at large and is unrelated to coal workers' pneumoconiosis in anyway. He also did not have clear documentation of pulmonary emboli. This man clearly would have expired at the same time and for the same reason regardless of his occupational exposure and occupational history.

(EX 3).

Dr. Abdul K. Dahhan performed a record review on behalf of the Employer and issued a report on August 13, 1998. His conclusions and reasoning were that the Miner had radiological evidence consistent with simple coal workers' pneumoconiosis; that he "died as a result of pneumonia and pulmonary emboli, conditions that are

not caused by, contributed to or aggravated by coal dust exposure or occupational pneumoconiosis"; and that he had no evidence of complicated CWP as the chest X-ray was negative for same and the more-sensitive CT scan failed to confirm the possibility of complicated CWP. (EX 5). Dr. Dahhan is board-certified in internal and pulmonary medicine. (EX 6).

Dr. Wheeler was deposed on September 21, 1998. He testified as to the make-up of the ILO-U/C forms for interpreting X-rays. He stated that "we don't want to call [a mass] a large opacity when we're actually dealing with a cancer -- active tuberculosis process or active infection because a worker can be signed out as having a large silicotic opacity." He implied that the worker would then not be on guard for cancer or infections. When asked whether he categorized any of the masses or densities on the Miner's X-rays as a large opacity, Dr. Wheeler said that "[n]o, there was no background nodularity or small nodules."

When questioned about the April 23, 1996 CT scan, Dr. Wheeler stated that he found "minimal nonspecific interstitial fibrosis mainly in the right mid lung and lateral periphery of the left mid lung, and there are a few tiny nodules in the periphery of the left lower lung and pleura, and all compatible with granulomatous disease." He was certain that the changes were not of CWP because CWP "would give symmetrical nodules generally in the central portion of the mid and upper lungs. When it goes to the periphery, it's always going to be predominating, in my experience, centrally in the mid lungs." Due to the stability of the masses between 1986 and 1996, Dr. Wheeler does not think that the Miner had cancer. As to his diagnosis of conglomerate tuberculosis and how likely it would have gone undetected, Dr. Wheeler stated that he did that think it was undetected as the doctors would have had to see the X-ray evidence of the masses. He continued that "[w]hether they treat him or not is dependent on whether they get organisms out. If they don't get organisms out and if he's not losing weight, they may not need to treat. If they were, they are getting organisms out, it would be malpractice not to treat him." He gave histoplasmosis as an alternative diagnosis. (EX 7).

Dr. Khokar was deposed on September 22, 1998. He testified that when he first saw the Miner he already had complicated pneumoconiosis, and that he had a "downhill course". He stated that the cause of the Miner's death was "[r]espiratory failure complicated by bilateral pneumonia and underlying disease of complicated pneumoconiosis, and massive pulmonary fibrosis."

Dr. Khokar testified that every time the Miner had an acute exacerbation of his condition, he obtained an X-ray and reviewed it with the radiologist. He stated that as an attending physician, he was, of course, aware of alternative causes for changes on the X-rays, but that he came to the conclusion of pneumoconiosis. He did not attribute the changes to tuberculosis because he observed over a long period that the Miner did not exhibit any of the signs of

tuberculosis nor have a history of tuberculosis. Because the Miner was on steroids, a PPD test would have been useless. Sarcoidosis was not believed to be a cause because the Miner did not have enlarged egg-shell type lymph nodes in the mediastinum, and he had no skin or eye lesions, spleen enlargement, or other feature of sarcoidosis. On April 18, 1997, the Miner expectorated some blood stained sputum, but cytology was negative for any tumor, so the expectorant was considered due to the inflammation in the bronchial tubes and not due to hemoptysis (actual blood spitting).

Dr. Khokar further testified that there was some background of simple pneumoconiosis, but that the x-rays in this case were so heavily involved that there was very little background overall. He did not find any evidence of cancer, and testified that cigarette smoking would cause some linear fibrosis, but not nodular fibrosis. (CX 2).

Dr. Hatahet was deposed on September 28, 1998. He testified that he saw the Miner in consultation during his final hospitalization. He performed a physical examination, and reviewed the X-ray and laboratory work. His diagnoses were decompensated respiratory failure, pneumonia of the right lung, significant tachycardia, and pre-existing fibrotic lung disease/complicated pneumoconiosis. The X-ray showed conglomerate masses in the upper lobes. Dr. Hatahet further testified that the combination of the pneumonia and fibrotic lung disease "deprived the patient of the majority of his functional lung." He ruled out cancer because of the masses remaining stable over a lengthy period of time and the lack of a smoking history. He explained why he did not reach a diagnosis of sarcoidosis or tuberculosis. As to pneumoconiosis being instrumental in causing the Miner's death, Dr. Hatahet testified that:

> The patient didn't have normal lung capacity when he presented with his final illness. He already had impaired lung function. Since there was no other medical process to explain that, in my mind at least, I believe that CWP had deprived him of a degree of his lung reserve with which he could have coped with the acute illness.
>
> Of course, he went on to have very severe disease, meaning the pneumonia and pulmonary emboli, both of which took the rest of his functional lung away, and he could not be kept alive even on the mechanical life support.

(CX 3).

Dr. Castle was also deposed on September 28, 1998. He explained that "[h]ilar adenopathy is a term that we use for enlargement of the lymph glands at the root of the lung or at the hila of the lung." He noted that "steroids are an immunosuppressive drug that reduces one's immunity to certain types of infection, and it appears that he developed evidence of a Serratia infection." He did not think that the Miner had

tuberculosis. His diagnosis was sarcoidosis. (EX 8).

Pneumoconiosis and Causation

The regulations provide four methods for finding the existence of pneumoconiosis: chest X-rays, autopsy or biopsy evidence, the presumptions in §§ 718.304, 718.305, and 718.306, and medical opinions finding the claimant has pneumoconiosis as defined in §718.201. See § 718.202(a)(1)-(4). In the instant case, there are chest X-rays, evidence of complicated pneumoconiosis pertinent to § 718.304, and medical opinions to consider. §§ 718.202(a)(1), (3), (4). There is no autopsy or biopsy evidence. §718.202(a)(2).

Beginning with the X-ray readings, Dr. Wheeler (BCR/B) found masses which he related to conglomerate tuberculosis. Although these masses met the size requirements to be categorized as large opacities in the ILO-U/C system, Dr. Wheeler did not mark them as such because he did not want a coal miner to mistakenly assume that the possibility of cancer or infection was excluded, and because he did not see any background nodularity. While there was interstitial nodularity and even some tiny nodules on the X-rays and CT scans, Dr. Wheeler determined that they were not consistent with CWP or silicosis because of their peripheral placement.

Dr. Castle (B) likewise did not relate the changes to CWP because he did not find small rounded regular opacities. He related the linear bilateral infiltrates, hilar adenopathy, and possible hilar mass to sarcoidosis. Dr. Castle reviewed both X-rays and the CT scans.

Dr. Fino (B) read X-rays and the CT scan as showing diffuse alveolar filling disease process. He found no rounded opacities. He noted enlargment of the hilar lymph glands.

Dr. Dahhan (B), on the other hand, related the changes to pneumoconiosis. He found rounded opacities (p and q/p). He noted questionable coalescing ("ax") of the pneumoconiotic nodules. He questioned whether the mass represented cancer.

Dr. Aycoth (BCR/B) found the changes to be consistent with pneumoconiosis, as did Dr. Gaziano (B). They found mixed and rounded opacities (p/s and p), respectively.

Dr. Pope found pneumoconiosis and bullous change.

Dr. Shahan, Fowler, and Rao all read X-rays in the course of the Miner's treatment. Dr. Shahan found chronic severe interstitial disease and bilateral perihilar soft tissue masses, including one conglomerate mass, and midlung pneumonia. Dr. Fowler found right midlung pneumonia, chroinc interstitial fibrotic scarring, and COPD. Dr. Rao found right midlung pneumonia, and conglomerate masses of pneumoconiosis.

As to the medical opinions, Dr. Vasudevan, who examined the

Miner in 1987, diagnosed category 1 pneumoconiosis by X-ray.

Dr. Abernathy (BCIM) also examined the Miner in 1987, and diagnosed CWP and bullous emphysema related to coal dust exposure. He found severe pulmonary disease, with desaturation of the blood on exercise.

Dr. Qazi's 1988 examination resulted in a diagnosis of significant COPD due to coal dust exposure and interstitial pneumoconiosis.

Dr. Morgan (BCIM) reviewed the medical records in 1988, including the only three X-ray readings obtained to that point, and accepted those readings of pneumoconiosis.

Dr. Fino (BCIM/PD) likewise accepted the readings of pneumoconiosis in 1988, but changed his opinion in 1998 based on new X-ray readings. He concluded that the changes were consistent with sarcoidosis, not pneumoconiosis. His reasons were that the use of bronchodilators was inconsistent with CWP, and CWP cannot cause significant obstruction. He did not relate the masses to complicated pneumoconiosis because of his determination that there was no background of simple CWP and because the masses were located in the lower zones.

Dr. Khokar (BCIM & treating physician) examined the Miner on numerous occasions from 1993 until his death in 1997. His diagnoses were COPD, complicated pneumoconiosis, and massive pulmonary fibrosis. He treated the Miner with bronchodilators and with steroids.

Dr. Krishnan saw the Miner in consultation while he was hospitalized in 1996. He noted physical examination findings consistent with pulmonary fibrosis. His diagnoses were COPD and CWP.

Dr. Sherer treated the Miner on one occasion in 1997. His impression was advanced obstructive lung disease.

The ventilation test in 1997 showed severe obstructive airway disease bilaterally.

Dr. Gaziano (BCIM/PD) reviewed medical records, and concluded that CWP was a significant contributor to the Miner's death. He did not find any complicated pneumoconiosis, but he noted that he only had the X-ray readings of were made more than ten years earlier.

Dr. Hatahet (BCIM) treated the Miner during his last hospitalization. He diagnosed advanced CWP, finding conglomerate masses in the upper lobes.

Dr. Castle (BCIM/PD) reviewed medical records and reached a conclusion of sarcoidosis. He did not attribute any of the changes to pneumoconiosis, noting that the Miner had minimal abnormalities in 1987 after leaving the mines.

Dr. Dahhan (BCIM/PD) reviewed medical records and concluded that the Miner had simple CWP, but not complicated CWP.

Weighing all this evidence, I find that the X-rays presented changes which were consistent with pneumoconiosis, regardless of whether a particular reader wanted to classify irregular opacities otherwise. The ILO-U/C form provides for the rating of irregular opacities. It is not limited to round, regular opacities. Therefore, I find the X-rays to be positive for changes consistent with simple pneumoconiosis under § 718.202(a)(1), and the Claimant is entitled to invocation of the rebuttable presumption that the pneumoconiosis arose from the Miner's coal mine employment.

Dr. Castle's conclusion of no CWP due to minimal abnormalities in 1987 is not clearly supported by a comparison of the X-ray readings and, in any event, his reasoning runs counter to the Act's recognition that pneumoconiosis is "a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure." I therefore find that Dr. Castle's opinion does not rebut the presumption.

Dr. Fino stated that CWP cannot cause significant obstruction, and that the use of bronchodilators is inconsistent with CWP. To the extent Dr. Fino’s opinion is based upon the presence of an obstructive defect, I find this his reasoning is inconsistent with the Act. Further, the fact that a bronchodilators may be ineffective for the treatment of CWP does not mean they are contraindicated or that a physician who would prescribe them would be engaged in inappropriate treatment. Use of bronchodilators to treat an ailment is not sufficient to rebut the presumption. I therefore find that Dr. Fino's opinion does not rebut the presumption.

Drs. Khokar and Hatahet both gave credible and convincing testimony at their depositions that the disease process the Miner had was pneumoconiosis. Dr. Khokar explained how other differential diagnoses, particularly cancer, tuberculosis, and sarcoidosis, were excluded. In fact, all the physicians are now in agreement that the Miner did not have cancer, as certain as any physician can be with this evidence. Dr. Khokar's testimony shows that the nature of his relationship with the Miner as his treating physician, the four years duration of that relationship until the Miner's death, the frequency of his examinations in and out of the hospital, and the extent of his treatment of the Miner, puts him in the best position to state what disease process the Miner had. 20 C.F.R. § 718.104(d)(1)-(4).

All of the X-rays which were classified as positive for simple pneumoconiosis, showed the changes to be present in all six zones or in the upper and mid zones, and that the opacities were completely or mainly round. None of the CT scans were stated as positive for simple pneumoconiosis, but the reasoning of the readers on the CT scans is no different from their reasoning on the X-rays, and the latter has already been dismissed.

I therefore find that the Miner's pneumoconiosis arose from

his coal mine employment. 20 C.F.R. § 718.203(b).

All but one of the examining physicians diagnosed coal workers' pneumoconiosis as it is defined clinically and/or legally. The one physician who did not, Dr. Sherer, diagnosed advanced obstructive lung disease and did not address the cause. His opinion does not refute the others' findings. Their opinions are documented and well-reasoned. I therefore find that coal workers' pneumoconiosis is also established under § 718.202(a)(4).

As to complicated pneumoconiosis, the masses certainly met the size requirement of large opacities as set forth in the deposition testimonies of Drs. Wheeler, Khokar, and Hatahet. The "OD" section is the place where a physician indicates the possibility of cancer and recommends that the patient's regular physician be notified, and I find Dr. Wheeler's concern about a miner being misled to be baseless reason for not checking off the large opacity section. The argument that the masses present absent a background of simple pneumoconiosis, and therefore cannot be complicated pneumoconiosis, is unsubstantiated given the findings under §§718.202(a)(1), 718.203(b).

While Dr. Wheeler opined that the masses were most likely representative of conglomerate tuberculosis, his deposition brought out the inability to make a diagnosis absent examination of the Miner or knowing his medical history. No other physician who examined the Miner or reviewed his medical history diagnosed tuberculosis. Therefore, I find Dr. Wheeler's suspicions are unsupported by the other evidence.

For these reasons, I find that the Claimant has established the presence of complicated pneumoconiosis under § 718.304(a) and is entitled to invocation of the irrebutable presumption of death due to pneumoconiosis. She is therefore entitled to benefits under the Act on this basis.

I also note that the evidence establishes that the Miner died a respiratory death and that his interstitial lung disease, which has been proven to be pneumoconiosis, played a substantial and significant role. While the use of steroids may have complicated his death, the steroids were prescribed by Dr. Khokar to treat the Miner's pulmonary condition, and as a consequence, pneumoconiosis and the treatment of it played a role in his death. The steroids apparently made the Miner more susceptible to pneumonia.

Accordingly,

ORDER

IT IS ORDERED THAT the Employer CONSOLIDATION COAL COMPANY shall pay to the Claimant BETTY BAILEY, widow of THOMAS BAILEY, all benefits to which she is entitled to under the Act.

A

STUART A. LEVIN
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. §725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date of this decision, by filing a notice of appeal with the Benefits Review Board at P.O. Box 37601, Washington, D.C. 20013-7601. A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits, Frances Perkins Building, Room N-2117, 200 Constitution Ave, N.W., Washington, D.C. 20210.